BRAIN ABSCESS WITH PATHOLOGICAL OBSERVATIONS

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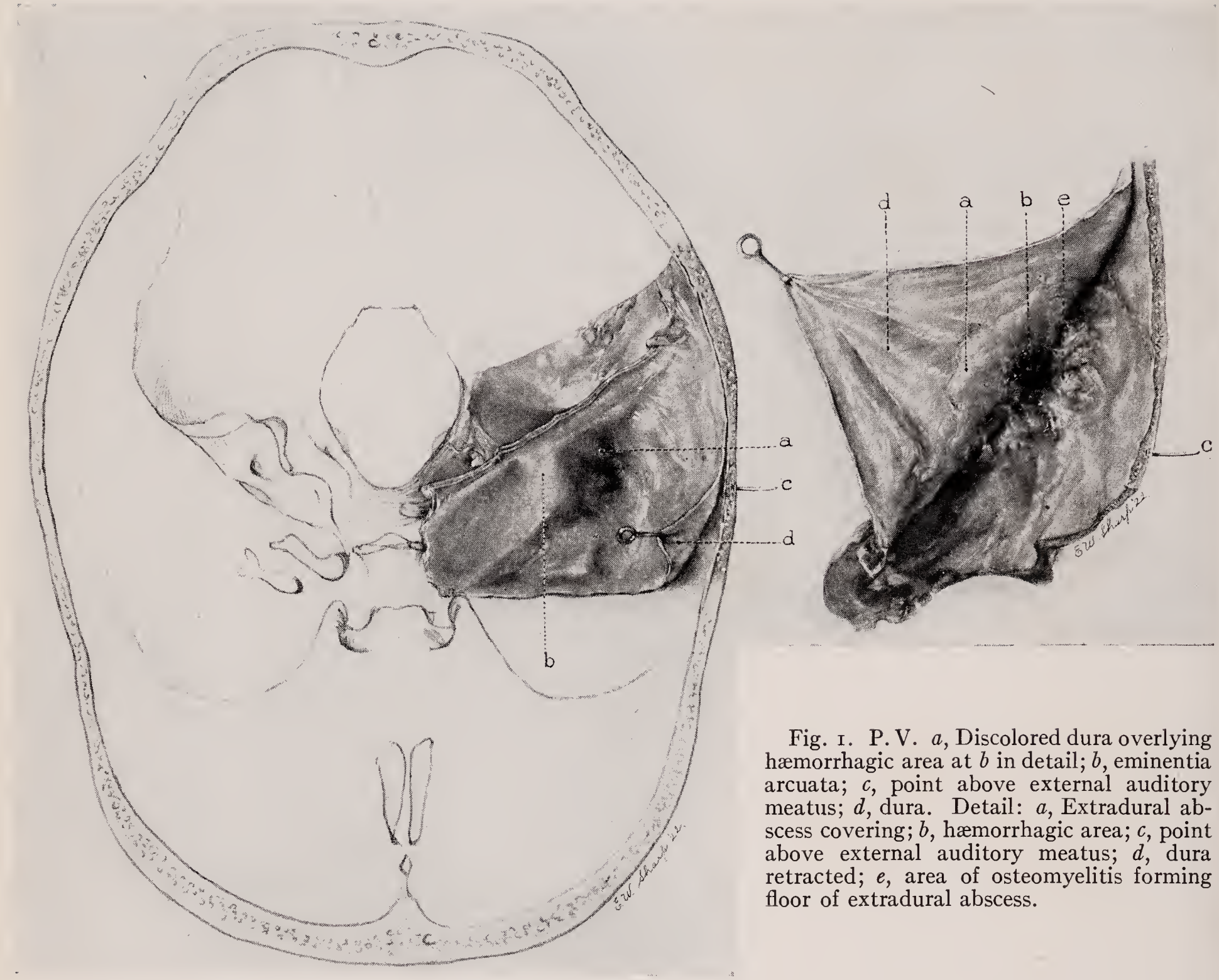

Fig. 1. P. V. *a*, Discolored dura overlying hæmorrhagic area at *b* in detail; *b*, eminentia arcuata; *c*, point above external auditory meatus; *d*, dura. Detail: *a*, Extradural abscess covering; *b*, hæmorrhagic area; *c*, point above external auditory meatus; *d*, dura retracted; *e*, area of osteomyelitis forming floor of extradural abscess.

Brain Abscess with Pathological Observations.—Charles Bagley, Jr.

BRAIN ABSCESS WITH PATHOLOGICAL OBSERVATIONS[1]

By CHARLES BAGLEY, Jr., M.D., F.A.C.S., Baltimore

THE substance of these remarks is part of a study of twenty cases of brain abscess. Seventeen were operated on with a mortality of 47 per cent. Eight of the cases operated upon and three unoperated upon died and an autopsy was performed in eight of the eleven fatal cases. Points of interest are shown in the illustrations.

These illustrations have been selected for the purpose of showing: (1) some of the avenues of infection, particularly those through which the infection reaches the brain, of tympanic cavity inflammation, and of gunshot and traumatic injuries; (2) behavior of the brain with regard to the formation of the abscess wall after the introduction of infection. Case reports are not attempted, but a few clinical facts are added, that the pathological illustrations will not lack the value of a clinical background. Initials of patients are given that the material here may be connected with the full clinical history of the patient, which it is planned to publish later.

Avenues of Infection

The material illustrating avenues of infection has been arranged in four groups, some of which are subdivided:

Group I. Presence of an extradural extension of the primary focus, with (a) protrusion of the distended dura into the cranial cavity; (b) direct extension from the extradural abscess; (c) invasion from the extradural abscess along the blood vessels.

Group II. Secondary invasion of the brain along the blood vessels without extradural link.

Group III. Penetrating brain injury with infection by foreign body, deep, with or without stalk. (a) Path infected and open, hence long abscess stalk; (b) path healed, hence no abscess stalk.

Group IV. Abscess superficial and open, secondary to direct laceration and infection of brain tissue.

GROUP I.—PRESENCE OF AN EXTRADURAL EXTENSION OF THE PRIMARY FOCUS

In considering the extension of infection from the tympanic cavity and accessory nasal sinuses to the brain, the dura must be placed first in importance as a barrier. Osteomyelitis of the wall of any of the cavities adjacent to the dura is likely to result, if thorough drainage of the pus is not accomplished within a reasonable time. Further extension of the inflammation is prevented when the process reaches the dura, which, because of its fibrous architecture is capable of active proliferation. Because of this defensive reaction, the inflammation is limited to the extradural space for a period of sufficient length to justify its designation as one of the stages of extension of infection from the primary abscess to the brain.

It is important that this stage be recognized clinically, because if the accumulation of pus is not evacuated early, further extension will

[1] A presentation of lantern slides shown before the Southern Surgical Association, December, 1922

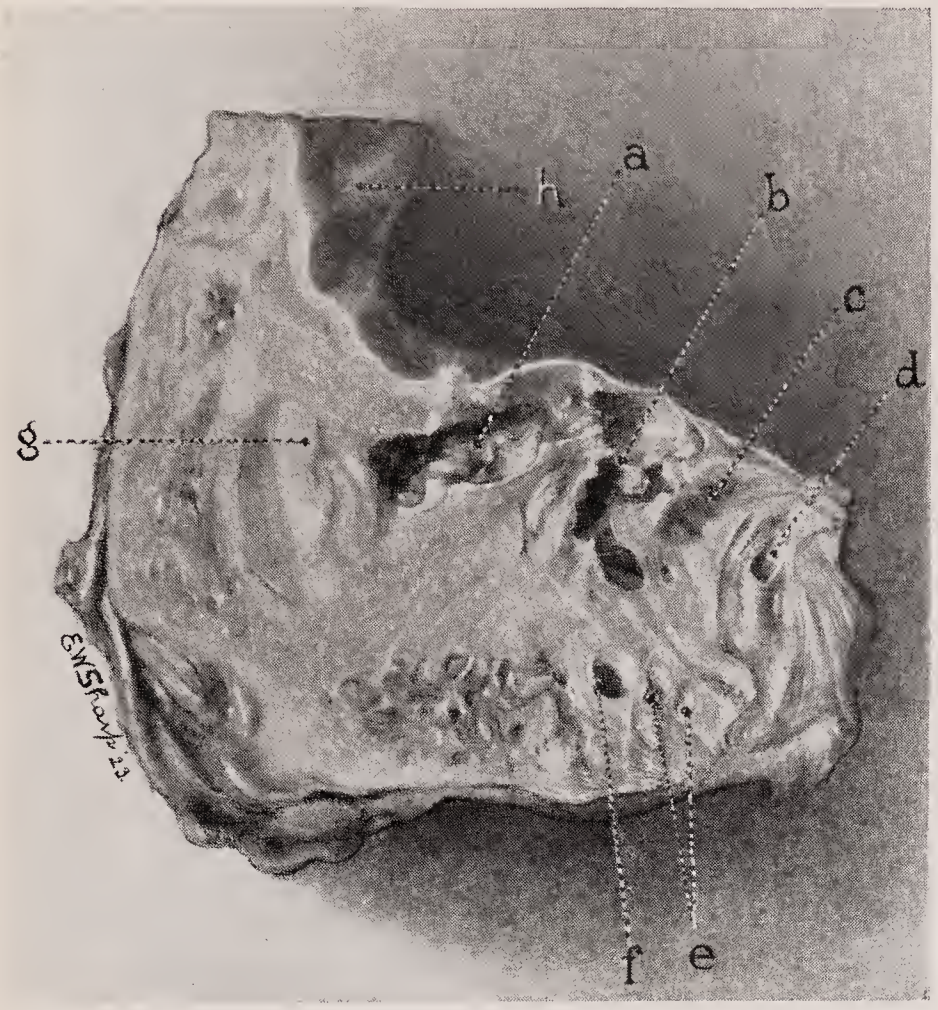

Fig. 2. P. V. A section through the temporal bone, shown in Figure 1. *a*, Mastoid antrum, seat of extensive suppuration, with osteomyelitis of its bony roof; *b*, tympanic cavity, also the seat of suppuration; *c*, hæmorrhagic extravasation; *d*, carotid artery; *e*, branches of the jugular vein; *f*, internal jugular vein; *g*, dura of the cerebellar fossa in close proximity to the suppurating antrum cavity; *h*, squamous portion of the temporal bone.

almost certainly occur. In no specimen of our material has this extradural accumulation been large, though several specimens demonstrate its occurrence. Extension from the extradural abscess in this series occurred in three different ways which have been arranged as subdivisions of Group I.

Fig. 3. R. L. Pedunculated dural abscess. *a*, Abscess stalk, point of attachment to dura; *b*, layer of cerebral tissue adherent to abscess; *c*, fibrous tissue wall of abscess; *x*, site of section shown in Figure 13; *y*, site of section shown in Figure 16.

The abscess shown in Figure 1 (frontispiece), a drawing of the temporal bone of P. V., may be considered typical of this extradural stage in the extension of the infection.

P. V., age 17. Left otitis media in early childhood, cured but purulent discharge from left ear again 3 weeks before admission. Left temporal lobe abscess for approximately 3 weeks. Meningitis, drainage of the abscess. Death.

This small extradural abscess was the result of necrosis of the roof of the tympanic cavity. The intracranial surface of the dura was not involved in the inflammation and there were no adhesions between the dura and the cortex. A cross section of this temporal bone (Figure 2) showed extensive suppuration of the tympanic cavity and antrum with osteomyelitis of their bony covering.

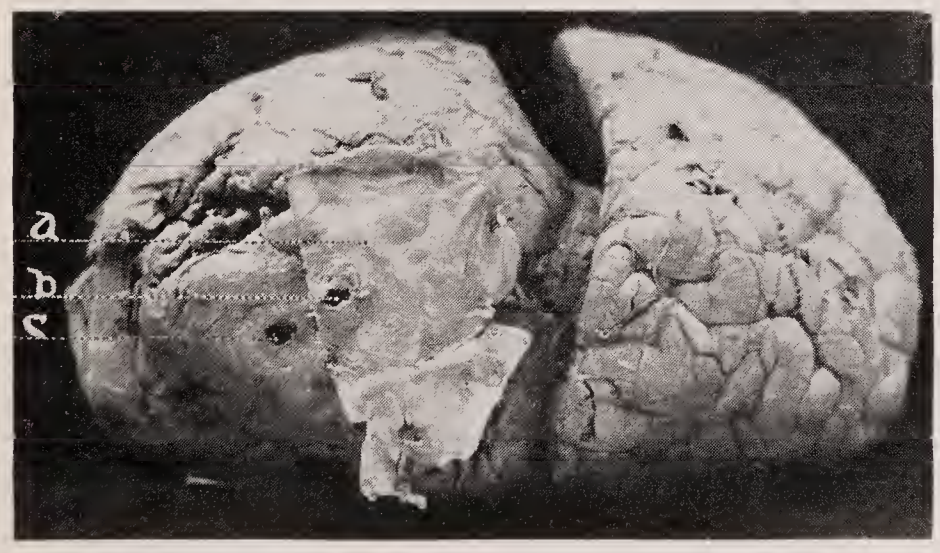

Fig. 4. L. F. Frontal view of brain with large abscess in right frontal lobe. *a*, Adherent dura of frontal lobe reflected toward mid line; *b*, perforation of dura; *c*, perforation in frontal lobe, which was continuous with *b* and formed the abscess stalk.

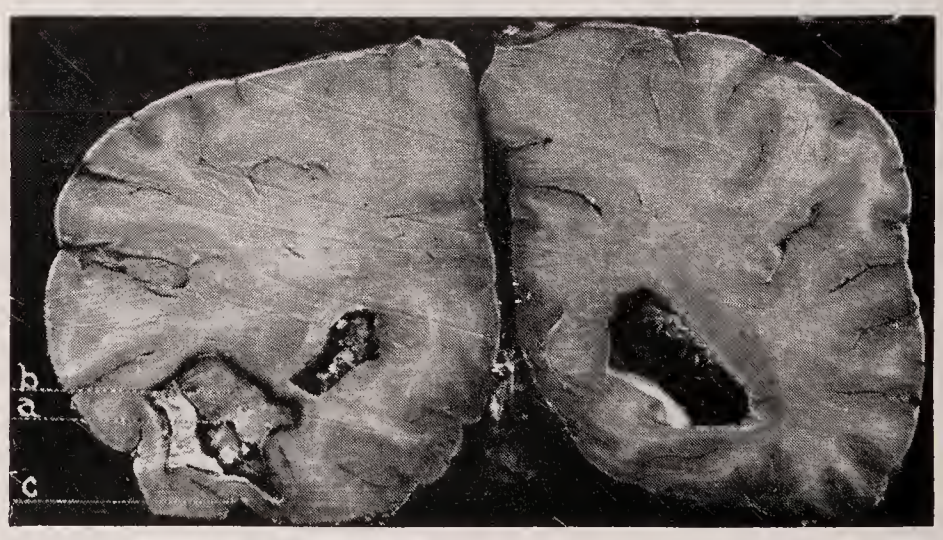

Fig. 5. P. V. Frontal section of a brain with left temporal lobe abscess. *a*, Abscess cavity; *b*, abscess wall and site of section shown in Figure 18; *c*, site of section shown in Figure 19.

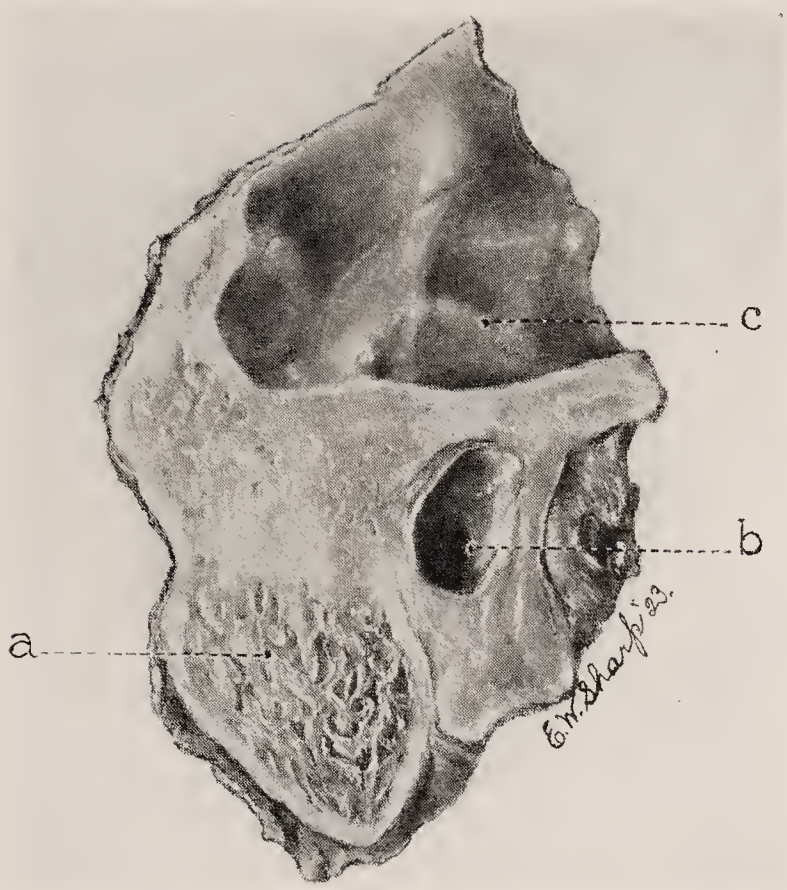

Fig. 6. P. V. A section through the temporal bone in Figure 1. *a*, Mastoid process, no evidence of suppuration; *b*, external auditory canal; *c*, squamous portion of temporal bone.

The specimen shown in Figure 3 was the result of extension by protrusion of the distended dura into the cranial cavity.

R. L., age 27. Pedunculated dural abscess, duration of approximately 4 months. Abscess removed without rupture. Recovery.

This abscess developed at the site of a necrosis of the occipital bone. The cause of the necrosis was not determined. The formation of the abscess differed from the usual extradural abscess in which the dura is merely depressed into the skull cavity. In this instance only a limited part of the dura pro-

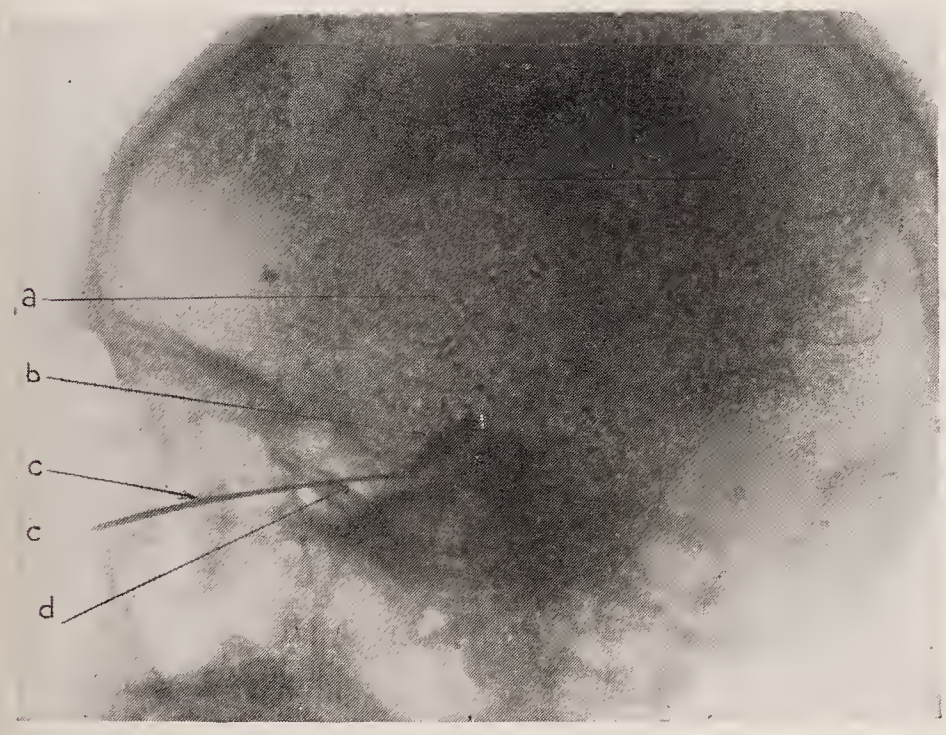

Fig. 8. E. M. *a*, Bone defect at site of subtemporal decompression; *b*, Metallic foreign body in left temporal lobe; *c*, probe passed through sinus marking tract through which foreign body passed; *d*, floor of middle fossa of skull.

Fig. 7. R. W. Upper surface of cerebellum, with abscess in left hemisphere underlying *b*. A cross-section of the abscess is shown in Figure 24. *a*, Point of spontaneous evacuation of abscess into posterior fossa.

liferated and was distended like a flask, owing to the slowness of the accumulation of pus.

Direct extension from the dural abscess is shown in Figure 4.

L. F., age 20. Influenza 4 months before admission, followed by right frontal sinus inflammation. Right frontal lobe abscess; probable duration 2 months. Drainage of abscess. Death.

After necrosis of the posterior wall of the frontal sinus, a condition revealed at operation, there must have been an extradural abscess as shown in Figure 1. The center of the inflammatory area of the dura was broken down and there was a direct communication between the extradural abscess and the frontal lobe, evidently through a local adhesion between the dura and the frontal lobe preventing the spreading into a general meningitis. The perforations shown at *b* and *c* formed the stalk of a large frontal lobe abscess.

An abscess, the result of invasion from an extradural abscess along the blood vessels, is shown in Figure 5, marking the further extension from the extradural abscess shown in

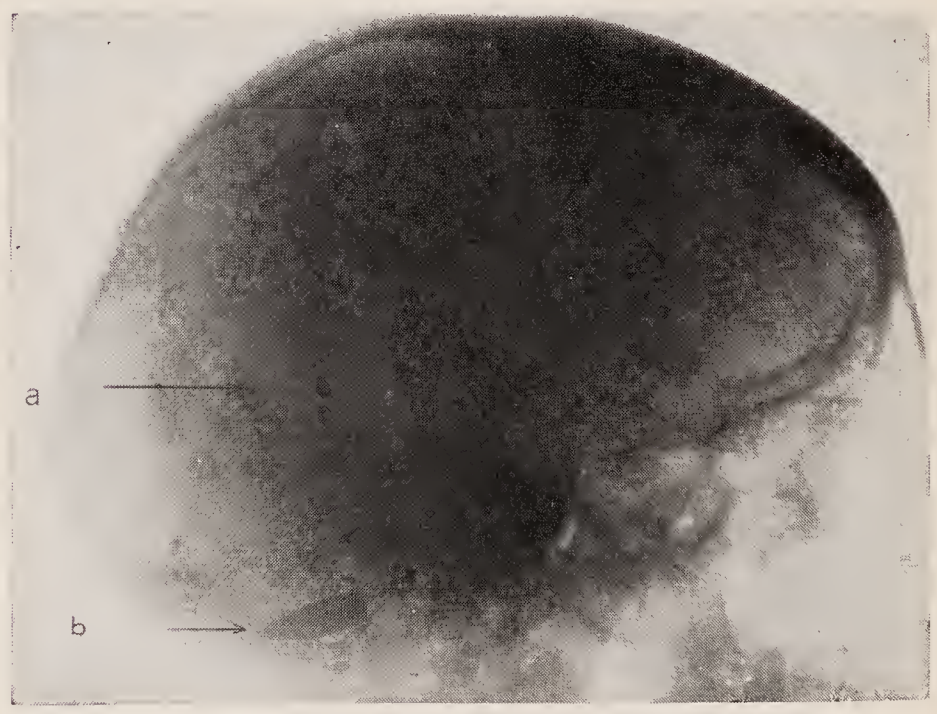

Fig. 9. P. L. *a*, Small skull defect, site of entrance of machine gun bullet seen at *b*.

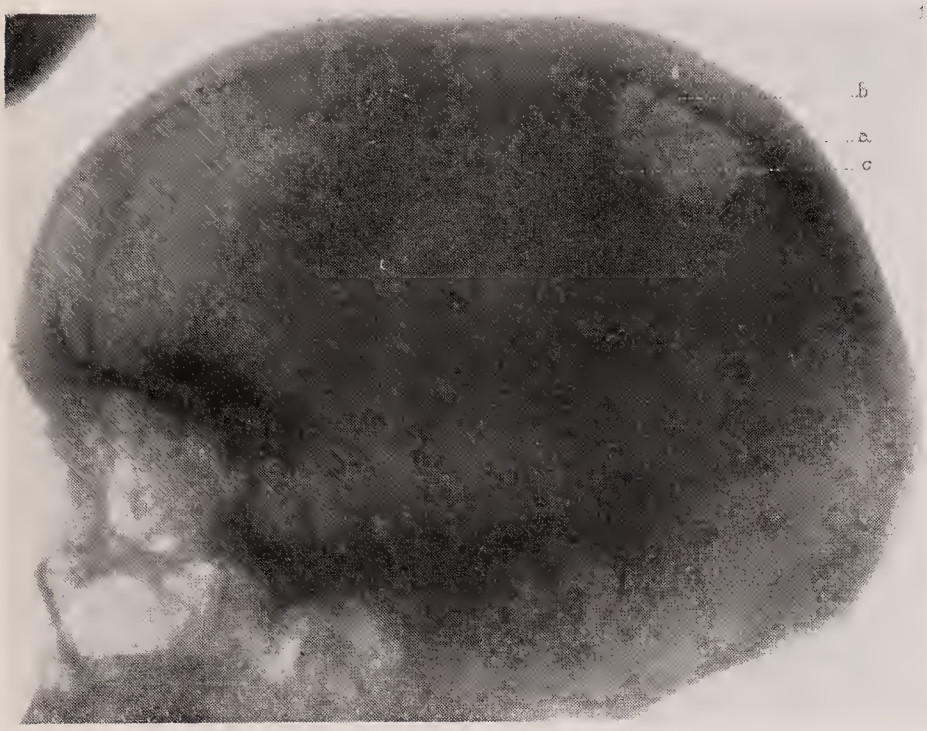

Fig. 10. W. F. M. *a*, Skull defect, resulting from gunshot injury; *b*, large indriven bone fragments; *c*, small bone fragments, the center of an abscess.

Figure 1. The meninges between this temporal abscess and the extradural abscess were

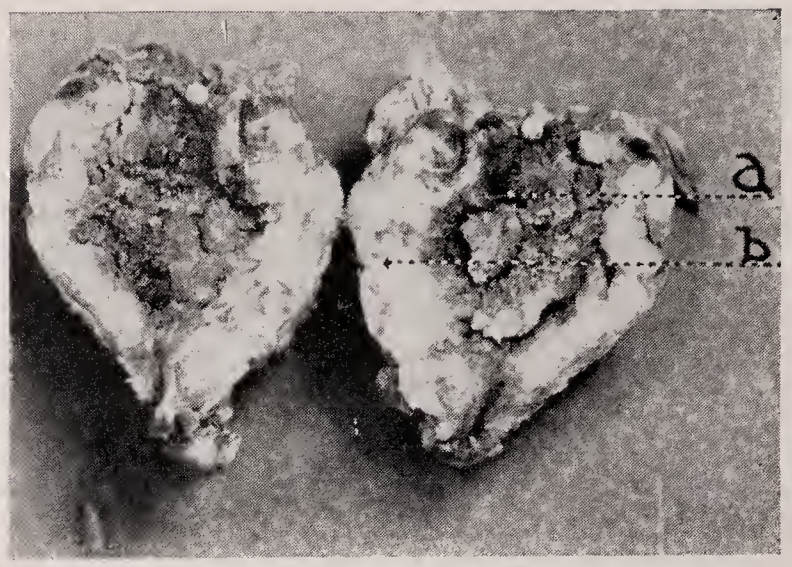

Fig. 11. W. F. M. *a*, Bone fragments shown at *c* in Figure 10; *b*, fibrous tissue abscess wall and site of section shown in Figure 12.

entirely free from inflammatory reaction. From the extradural abscess, or perhaps from the suppurating bone cavity, the inflammation seems to have been carried (as described under Group II) along a vein and the superior petrosal sinus into the substance of the temporal lobe.

The formation of an extradural abscess as a first link of cerebral infection must be fully appreciated, as adequate treatment of the primary lesion at this stage may prevent the infection from extending to the cerebral tissue. Extensive suppuration of the tympanic cavity and mastoid antrum with osteomyelitis of their bony roof occurs in chronic cases without involvement of the mastoid process, so that symptoms of mastoiditis need not occur; for example in Figure 6, the mastoid process of the temporal bone shown in Figure 1 is free from suppuration. Had symptoms of mastoid involvement appeared, the process would have been opened and the suppurating cavity in the bone, and perhaps the extradural abscess, adequately drained. Such a course was pursued with satisfactory results in the following case:

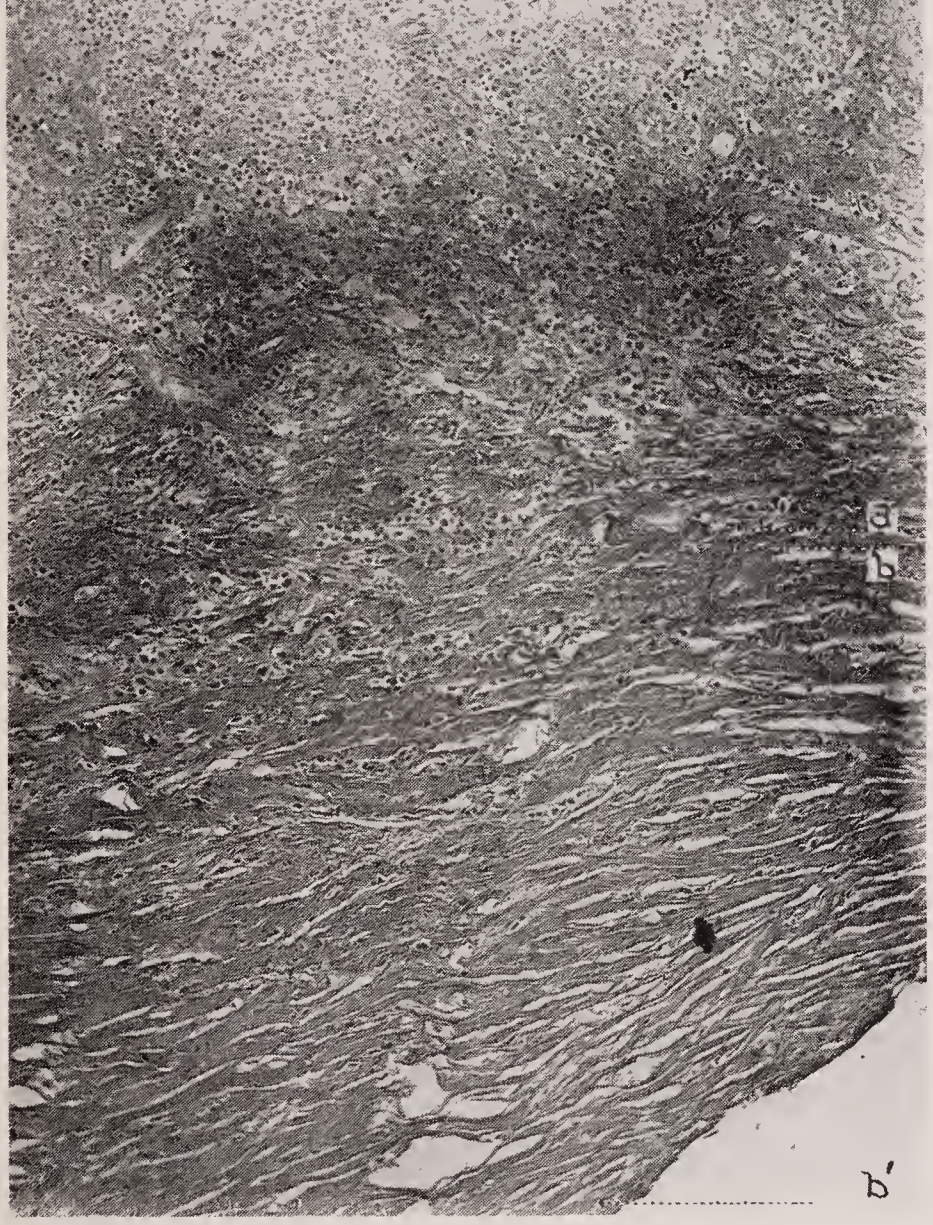

Fig. 12. W. F. M. Section of wall from *b* in Figure 11. *a* to a^1, Inner layer of abscess wall, showing young fibrous tissue elements; *b* to b^1, outer layer of abscess wall, showing adult fibrous tissue. ×85.

J. A. S., age 38, had purulent discharge from the left ear since his twelfth year. Headache and dizziness were troublesome for 6 months before operation. A mastoid operation revealed extensive necrosis in the neighborhood of the antrum, and after the operation there was free drainage of pus from above the antrum. The postoperative course was satisfactory until the eleventh day when there was frontal headache with mental confusion, slight aphasia, occasional twitching of the right shoulder girdle muscles, and gradually increasing stupor. As an exploring needle introduced into the left temporal lobe did not reveal pus, it was

Fig. 13. R. L. Section at *x* of wall of abscess shown in Figure 3. *a*, Abscess cavity; *b*, necrotic tissue covering inner surface of abscess wall; *c*, abscess wall shown also in Figure 14; *d*, brain tissue, the site of active glial proliferation. ×15.

Fig. 14. R. L. Higher magnification of a section from *c* in Figure 13. *a*, Adult fibrous tissue strands; *b*, young fibrous tissue elements. ×325.

concluded that the cerebral symptoms were due to œdema secondary to the extradural abscess rather than to an extension of the infection. A decompression was done—the value of which lay in the relief of intracranial pressure—while the inflammatory process was checked by drainage of the extradural abscess through the mastoid antrum, with complete recovery.

External drainage of the area of bone necrosis, however, did not prevent the formation of the abscess shown in Figure 3. It is possible that the drainage diminished the amount of pus to be taken care of by the abscess wall to a point where the proliferation of fibrous tissue could keep pace with the distention.

It is probable that the inflammation extends directly from the extradural abscess more frequently than by the other methods described. The tract between the abscesses in some instances remains patent and offers a means of spontaneous evacuation of the brain abscess. In one case there was a history of chronic otitis media for 14 years, the drainage from the canal varying in amount. At times it was profuse, extending in a small stream from the canal and over the side of the face for a period of several hours. For 3 weeks prior to admission there had been very little drainage, but signs of serious increase of intracranial pressure terminated in respiratory collapse. While artificial respiration was being carried on preparatory to draining the abscess, there was a free discharge of foul pus from the canal, so that further evacuation was not undertaken.

In spite of this spontaneous drainage, death resulted from the serious medullary disturbance. At autopsy there was found a sinus extending from the tympanic cavity to a temporal lobe abscess.

GROUP II.—SECONDARY INVASION OF THE BRAIN ALONG THE BLOOD VESSELS WITHOUT EXTRADURAL LINK

The superior petrosal sinus, receiving the veins from both the tympanic cavity and the cortex of the temporal lobe, constitutes an indirect vascular link through which infection may extend. The lateral sinus may likewise form a link between the mastoid cavity and

Fig. 15. R. L. A higher magnification of *d* in Figure 13, showing neuroglia fibrils. ×325.

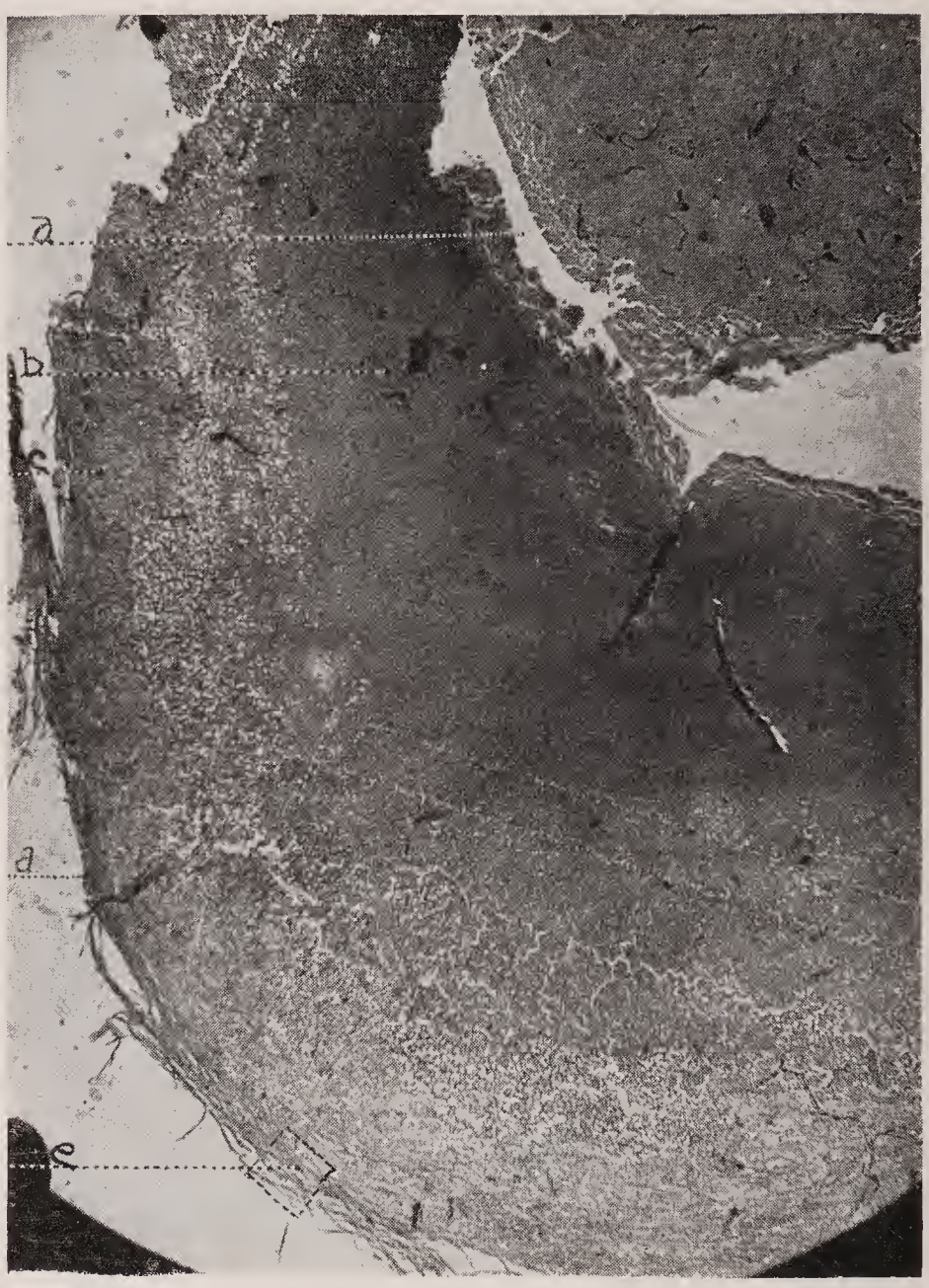

Fig. 16. R. L. Section at *y* of wall of abscess shown in Figure 3. *a*, Abscess cavity; *b*, necrotic substance covering inner surface of wall; *c*, young fibrous tissue elements; *d*, adult fibrous tissue; *e*, site of section shown in Figure 17. ×15.

the cerebellar hemisphere. The exact method of extension of infection along the blood vessels cannot be outlined except where there is a thrombosis of the sinus, in which case the infected content of the sinus may be dammed back into its tributaries, and thereby carry organisms into the poorly resistant cerebral tissue. It is probable that merely a condition of phlebitis with retardation of the blood current may cause this forcing back of infected material. The correctness of this explanation can be determined only by further observation; the facts being that abscesses occur in the temporal lobe and cerebellar hemisphere secondary to tympanic cavity and mastoid inflammation without visible connecting tracts, and that there exists the indirect anatomical connection stated above.

An illustration of an abscess formed in such manner is given in Figure 7.

R. W., age 15. Left otitis media, mastoiditis, and cerebellar hemisphere abscess. Probable duration of abscess 3 weeks. Streptococcus infection. Drainage. Death.

There is no evidence of involvement of the meninges, though the left cerebellar hemisphere contained a large abscess; the infection evidently having reached the deep substance of the cerebellum along the blood vessels.

GROUP III.—PENETRATING BRAIN INJURY WITH INFECTION BY FOREIGN BODY, DEEP, WITH OR WITHOUT STALK

Penetrating wounds, complete or partial, are prone to infection at any point along the tract. Experience in the front line hospitals proved the necessity of thorough cleansing of such wounds with a view to removing all devitalized tissue and foreign material, and when this was accomplished within a few hours after the injury, primary closure of the wound was possible. When the penetration was incomplete, foreign bodies were often lodged

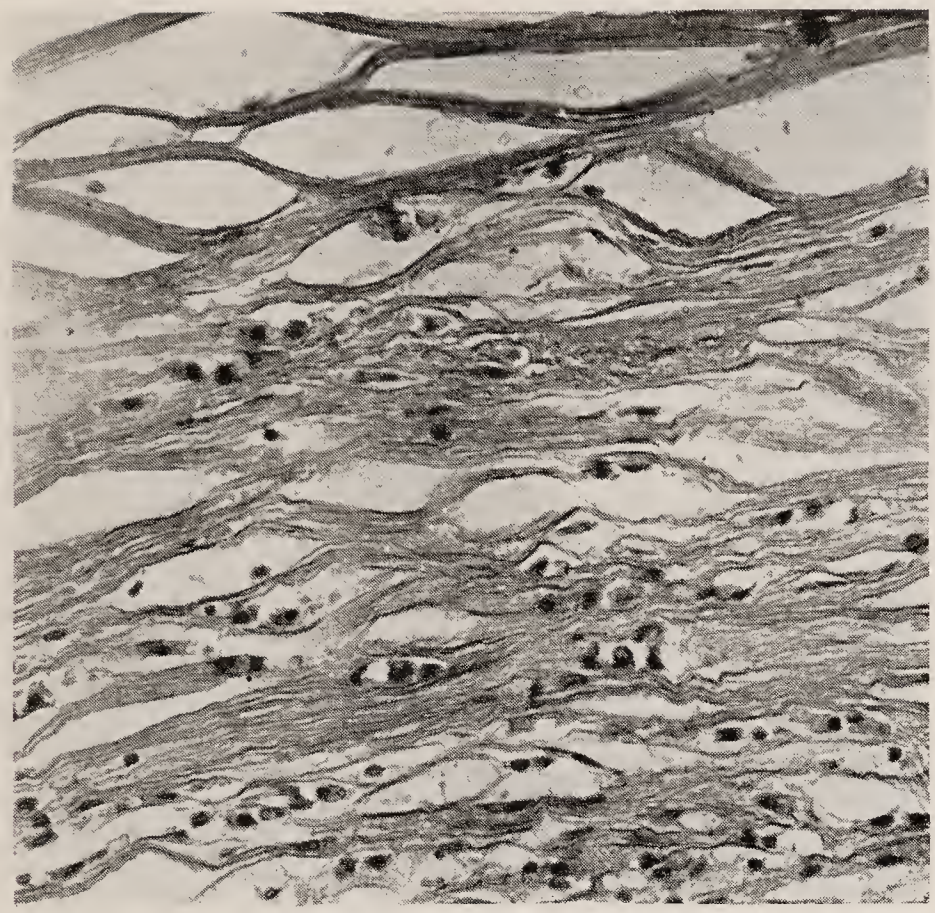

Fig. 17. R. L. Higher magnification of section at *e* in Figure 16 showing firm fibrous tissue strands. ×300.

at such a depth in the brain substance that their removal was not possible at the front line hospital. In addition, the stress of work at the front resulted in many cases being evacuated to the rear with incomplete operations. As a result of these conditions, cases were returned to this country, showing various types of cerebral lesions due to foreign bodies. In some, the foreign bodies were encapsulated and the healing was complete; in others, a discharging sinus extended from the foreign body in the substance of the brain to the skull surface, serving to prevent the accumulation of pus at the site of the foreign body; in others, the tract remained open but the formation of pus was in excess of the amount discharged through the sinus so that an abscess resulted; and in still others, the tract healed, and an abscess formed in the neighborhood of the foreign body.

An abscess with a path infected and open, forming a long abscess stalk, is shown in Figure 8.

E. M., age 25. Shell fragment entered temporal lobe through left malar region, August 17, 1918. Five months later symptoms of brain abscess. Left subtemporal decompression. Abscess drained, May 23, 1919. Recovery.

The tract through which the metallic foreign body entered the temporal lobe is shown in the X-ray photograph and is marked by the probe at *c*. This

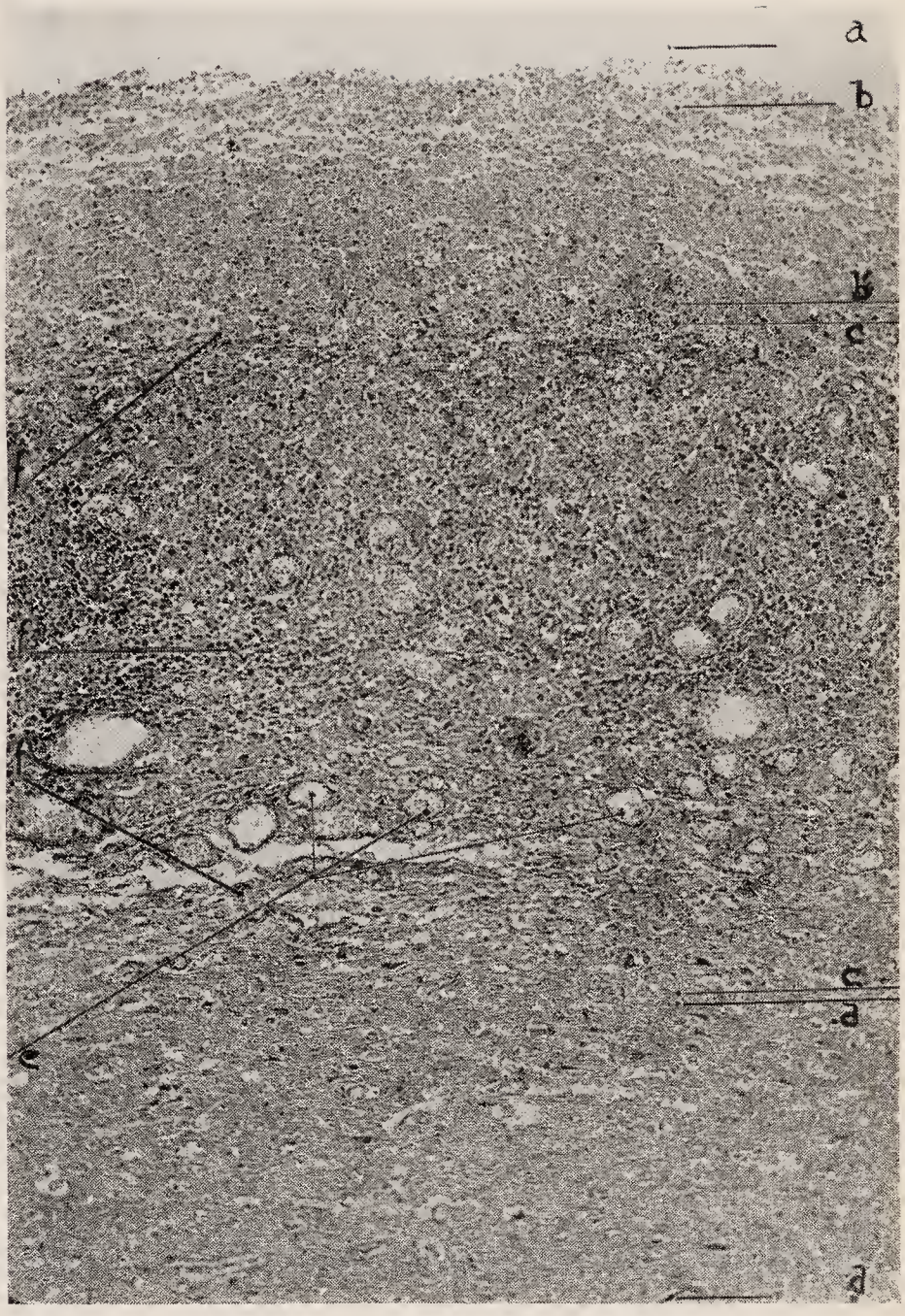

Fig. 18. P. V. A section from *c* in wall of abscess shown in Figure 5. *a*, Abscess cavity; *b* to b^1, necrotic substance on inner surface of abscess wall; *c* to c^1, abscess wall; *d* to d^1, brain tissue; *e*, thin wall blood vessels; *f*, framework of abscess wall consisting chiefly of fibrous tissue proliferated from the blood vessels. ×85.

tract remained open and continued to discharge pus from the abscess cavity from the time of the injury until the operation.

An abscess in which the path of the bullet healed, leaving no abscess stalk, is shown in Figure 9.

P. L., age 30. Wounded, September 27, 1918. Drainage of abscess and removal of bullet, May 10, 1919. Recovery.

The abscess cavity is cut off from the wound of entrance by healing of the tract which extended through the occipital lobe and tentorium into the right cerebellar hemisphere.

The discharging sinus in the case of E. M. no doubt prevented the formation of the temporal lobe abscess for a number of months, and would have been more effective but for the effort made to have this tract heal, as its connection with the foreign body was not

Fig. 19. P. V. A section from *b* in wall of abscess shown in Figure 5. *a* to a^1, Inner layer of abscess wall; *b* to b^1, hæmorrhagic extravasation in abscess wall; *c* to c^1, outer portion of abscess wall, showing extensive vascular proliferation and numerous punctate hæmorrhages. ×85.

considered. The abscess was finally drained through part of the original tract, for after its communication with the abscess was discovered, an incision in the temporal region exposed the proximal end of the tract just below the floor of the middle fossa of the skull, at which point very satisfactory drainage was obtained. The abscess was large, with extensive destruction of cerebral tissue, which resulted 3 years later in circulatory disturbance and impairment of function.

The other patient cited in this group was without symptoms of cerebellar disturbance. Removal of the foreign body was advised because of its size and the likelihood of cyst formation with destruction of cerebellar tissue. When the cerebellar cortex was opened there was a flow of pus which contained staphylococci.

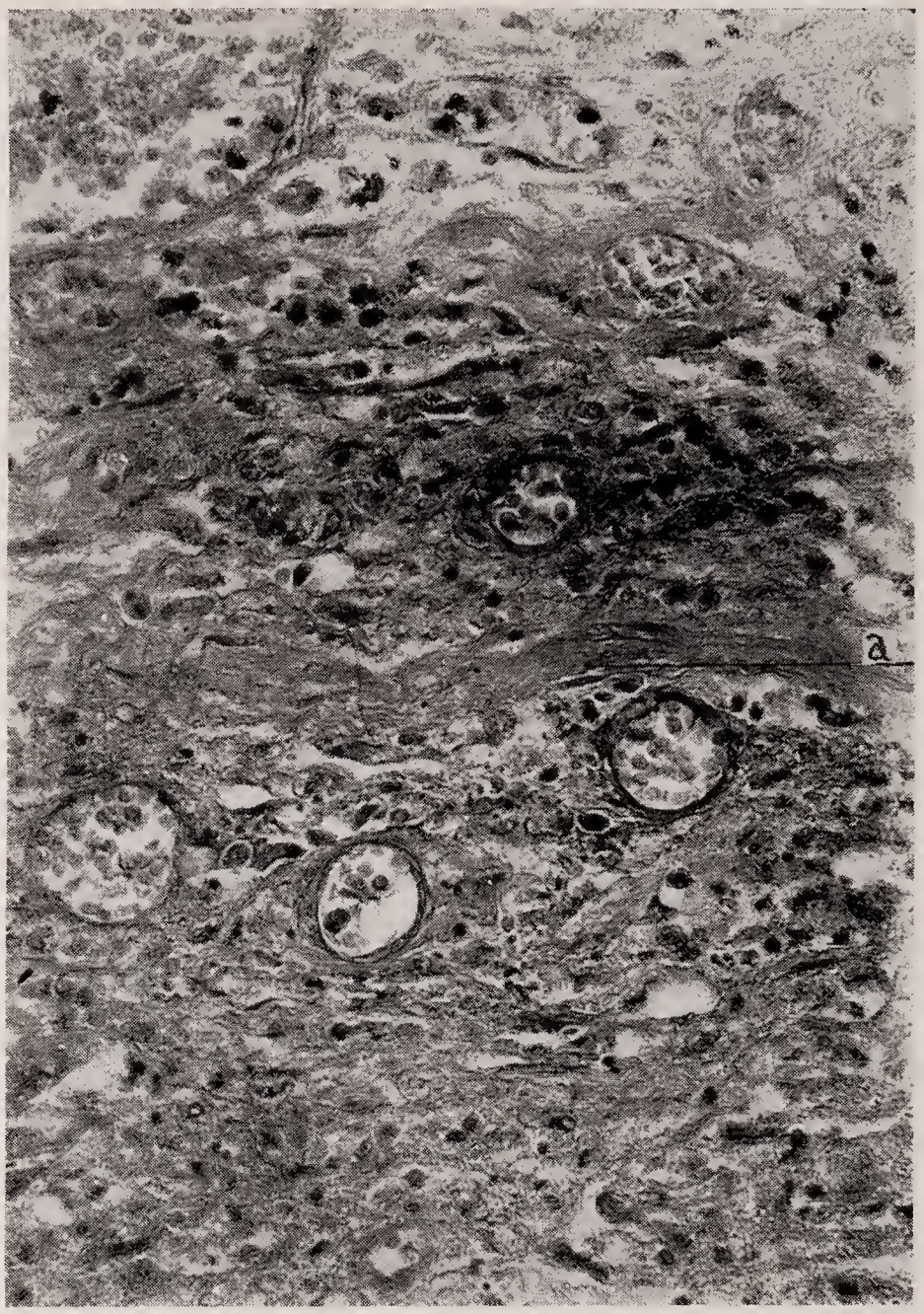

Fig. 20. W. F. M. Section from an abscess wall similar in type to that shown in Figure 18, but of longer duration. *a*, Small band of adult fibrous tissue. ×325.

GROUP IV.—ABSCESS SUPERFICIAL AND OPEN, SECONDARY TO DIRECT LACERATION AND INFECTION OF BRAIN TISSUE

Abscesses developing in neglected cases of compound fracture of the skull in which the surface opening is sufficiently large to permit a fairly free drainage of pus, often tend to do well when the foreign material is removed and drainage established. In these cases the encephalitis which follows the injury is localized and circumscribed by the proliferation of the neighboring mesoblastic tissue. Here again the dura plays an important rôle. In some cases at least the ragged dural flaps, dipping into the disorganized cortex, proliferate and completely shut off the foreign material from the brain, so that the resulting abscess is essentially extradural.

In the X-ray photograph in Figure 10 are seen shadows of the bone fragments extending in from the rim of the skull defect.

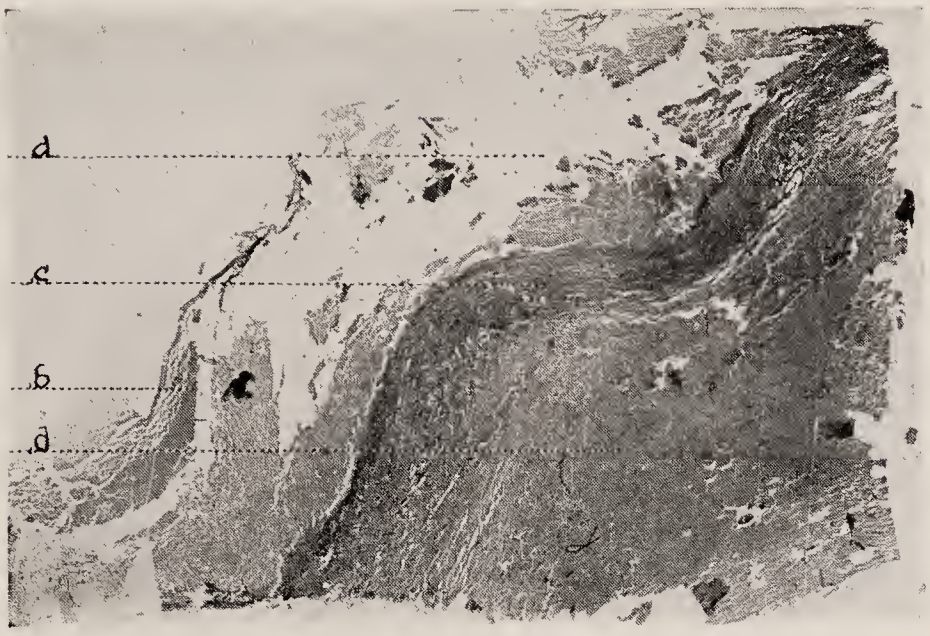

Fig. 21. L. F. A section from the wall of the abscess shown in Figure 4. *a*, Abscess cavity; *b*, disorganized tissue of inner abscess wall and site of section seen in Figure 23; *c*, abscess wall; *d*, brain tissue adjacent to the abscess wall. ×6.

W. F. M., age 25. Machine gun bullet wound, October 5, 1918. Bullet only removed at front line hospital. Wound unhealed; constant drainage of pus. Operation: Removal of firm wall abscess containing bone fragments, May 31, 1919. Death.

Fibrous tissue surrounding the large bone fragments at *b* formed the stalk of a thick walled abscess, in the center of which were contained the small fragments seen at *c*. The abscess is seen in Figure 11. The thick fibrous wall surrounding the fragments, as seen in Figure 10, must have been due to proliferation of fibrous tissue carried in with the bone fragments as the mass extended deep into the substance of the hemisphere entirely out of reach of any considerable amount of fibrous tissue. The character of the wall cannot be attributed to the long duration alone, as other abscesses in the hemisphere, the result of the same injury, showed almost complete lack of fibrous tissue elements.

Behavior of the Brain with Regard to the Formation of the Abscess Wall after the Introduction of Infection

The term abscess indicates a circumscribed accumulation of pus, and in this way the lesion under discussion, suppurative encephalitis, differs from the diffuse type of cerebral inflammation which is not amenable to surgical treatment. Only the end-results of the inflammatory process, namely, the abscess wall, will be treated in this paper. (1) The wall of the abscess is the most important factor in determining the outcome of well managed brain abscesses. As in all other inflammatory lesions the wall formation depends first upon the type of infecting organism, one of low virulence causing a more gradual accumulation of

Fig. 22. L. F. A higher magnification of wall of abscess seen in Figure 21 showing the membrane to consist of delicate fibrils. ×325.

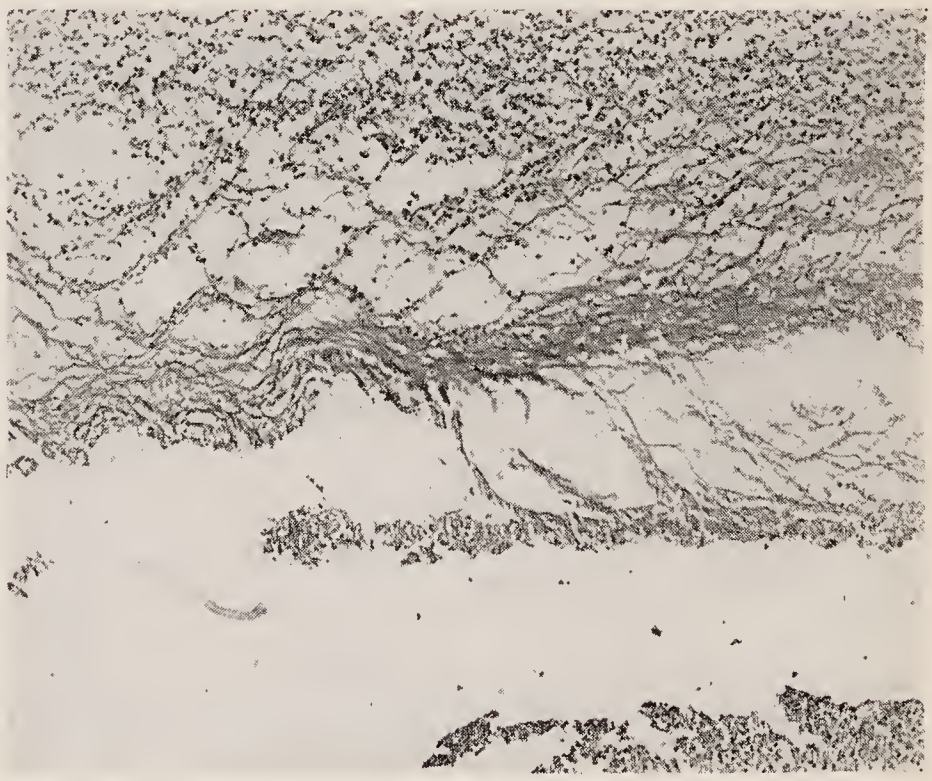

Fig. 23. L. F. Section of the innermost portion of abscess wall in Figure 21. Because of the necrosis the cellular elements have fallen out, leaving the delicate fibers in plain view. ×85.

Fig. 24. R. W. Cross section of cerebellum seen in Figure 7. *a*, Abscess cavity; *b*, area of hæmorrhagic extravasation, the result of thrombosis; *c*, site of section shown in Figure 25; *d*, site of section seen in Figure 26.

pus than one of greater virulence thus allowing sufficient time for the protective reaction of the tissue; (2) the resistance of the infected tissue is important, which protective reaction takes place principally in two kinds of tissue, fibrous mesoblastic and glial epiblastic. The fibrous tissue is far more effective but, unfortunately, is almost unavailable in the deep substance of the brain where glial tissue must suffice. In addition, it is influenced by the method of infection as shown in the first part of this paper.

Abscesses of long duration may have walls of greater thickness, but it is more likely that the duration is long and the wall thick because of the character of tissue available for proliferation.

TYPES OF ABSCESS WALL

Type I. Dense fibrous mesoblastic tissue wall.

Type II. Fairly firm wall, containing some fibers proliferated from neighboring mesoblastic tissue.

Type III. Walls of varying thickness the result of glial proliferation.

Type IV. Walls showing no evidence of a protective reaction.

Type I.—Dense Fibrous Mesoblastic Tissue Wall

If fibrous tissue is available for the abscess wall, it takes first place in the formation of the protective membrane. The meninges,

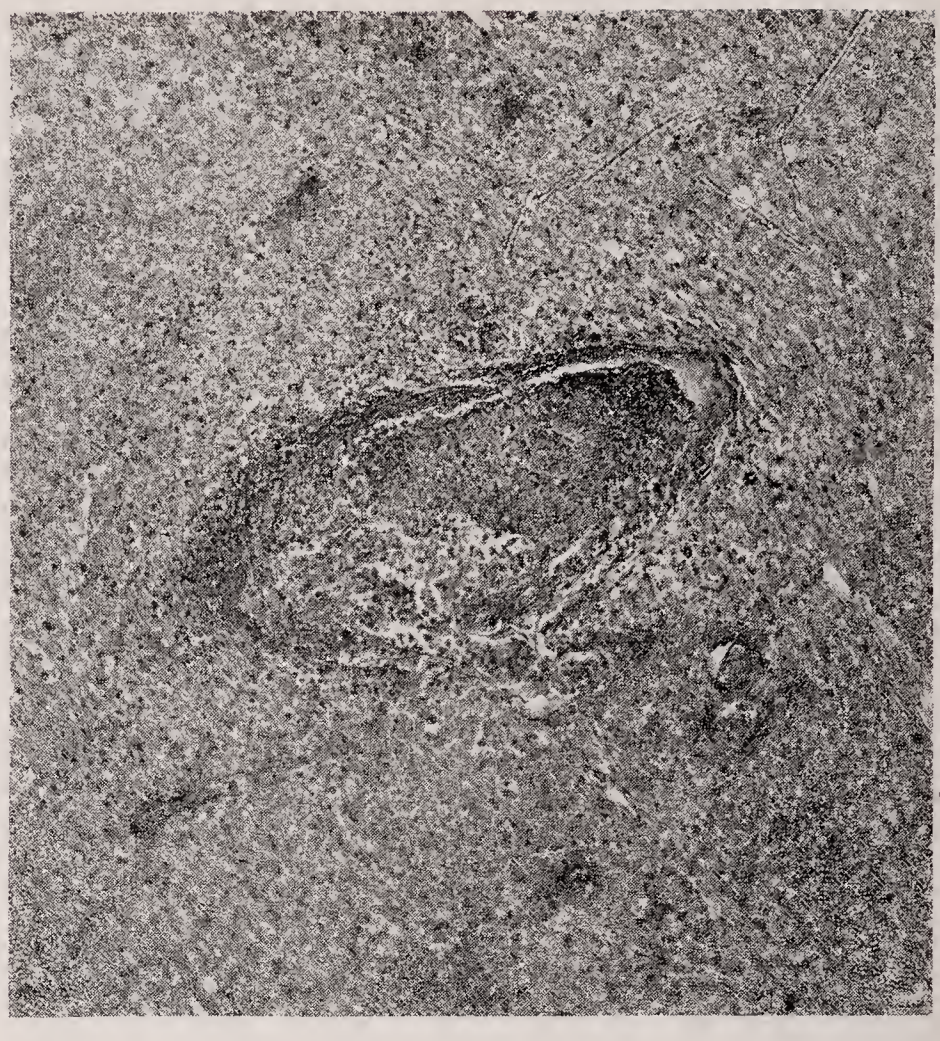

Fig. 25. R. W. Section from *c* in Figure 24. Note the large and small areas of thrombosis. ×85.

constituted largely of fibrous tissue, act as a barrier to pus (as in extradural abscess formation) and may furnish tissue for active proliferation and the walling-off of infection, even though the membranes be severely traumatized. Figures 11 and 12, for example, show the result of proliferation after a smashing skull injury. Figure 11 shows the firm wall abscess removed from W. F. M. in cross section. Figure 12, a photomicrograph, shows the wall to be made of mesoblastic fibrous tissue.

A very unusual reaction of the dura appears in Figures 12 to 17, photomicrographs of a large abscess of 4 months' duration, which was confined entirely within the limits of the dural tissue. In Figure 13, the same abscess as shown in Figure 3, the firm fibrous tissue wall was the result of proliferation of the slowly distending dura. The next figure, Figure 14, shows adult fibrous tissue strands and young fibrous tissue elements in this same abscess. Figure 16 shows the tensile quality of the fibers constituting the wall of the abscess, for a few strands were sufficient to protect the abscess against rupture. The quality of these strands is shown in Figure 17. Beyond the fibrous tissue wall there was neuroglial proliferation as shown in Figure 15. This latter

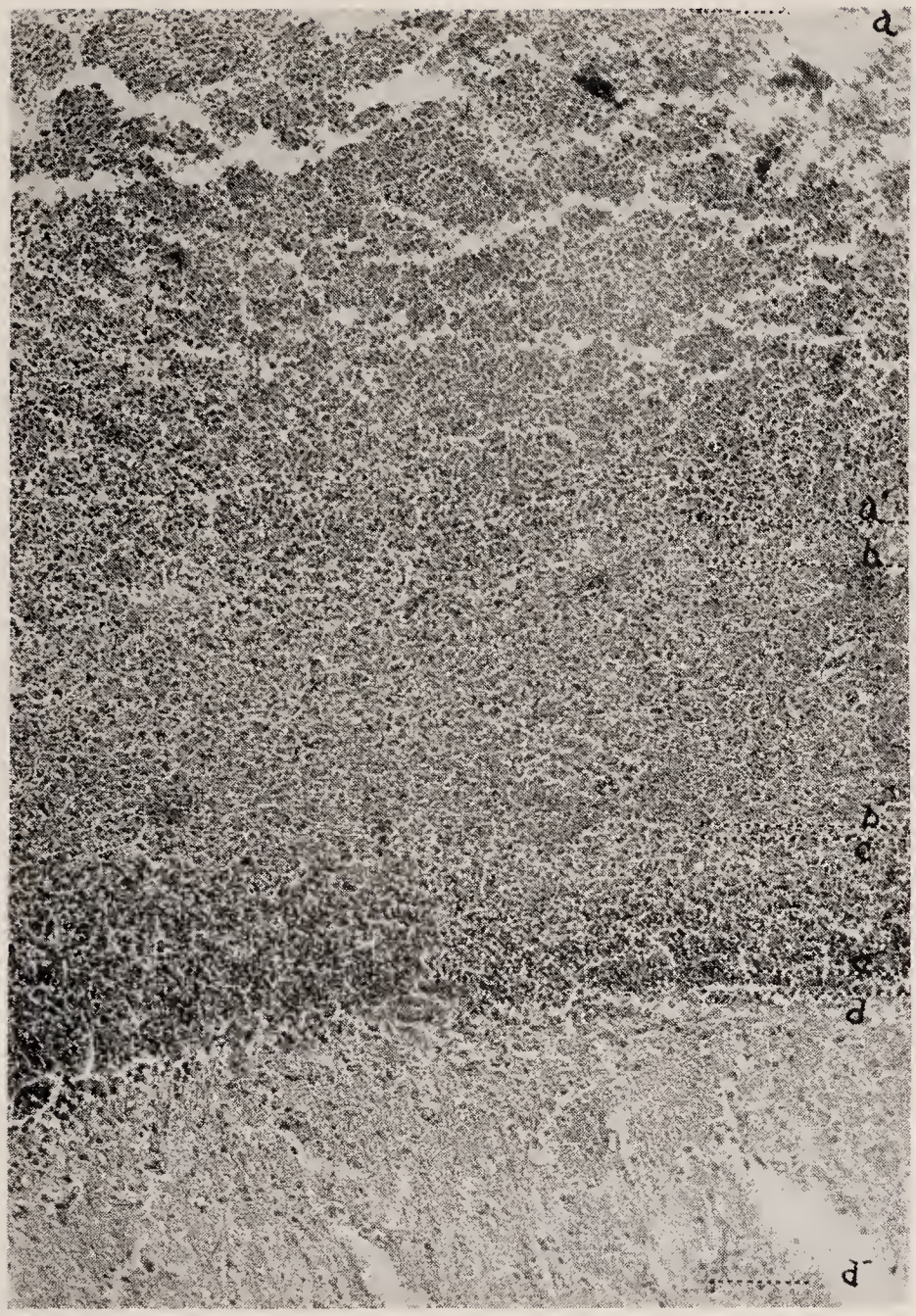

Fig. 26. R. W. Section from *d* in Figure 24. *a* to a^1, Necrotic tissue surrounding the abscess cavity; *b* to b^1, nervous tissue beyond the area of necrosis; *c* to c^1, layer of granular cells of the cerebellum; *d* to d^1, molecular layer of the cerebellum. ×85.

reaction of cerebral tissue, of little importance in this case, is the main protective reaction in the wall of the abscess designated as Type III in this paper. The similarity between Figure 15 and Figure 22 is striking.

It is evident that the method of infection and the propinquity of mesoblastic tissue to the site of infection influence greatly the above-described formation of an abscess wall.

Type II.—Fairly Firm Wall Containing Some Fibers Proliferated from Neighboring Mesoblastic Tissue

The type of abscess wall shown in Figure 18, while not the most valuable, represents the usual form of reaction when the infection occurs deeper than the fibrous tissue coverings. The chief reaction takes place in the glia, but this is augmented by proliferation from the

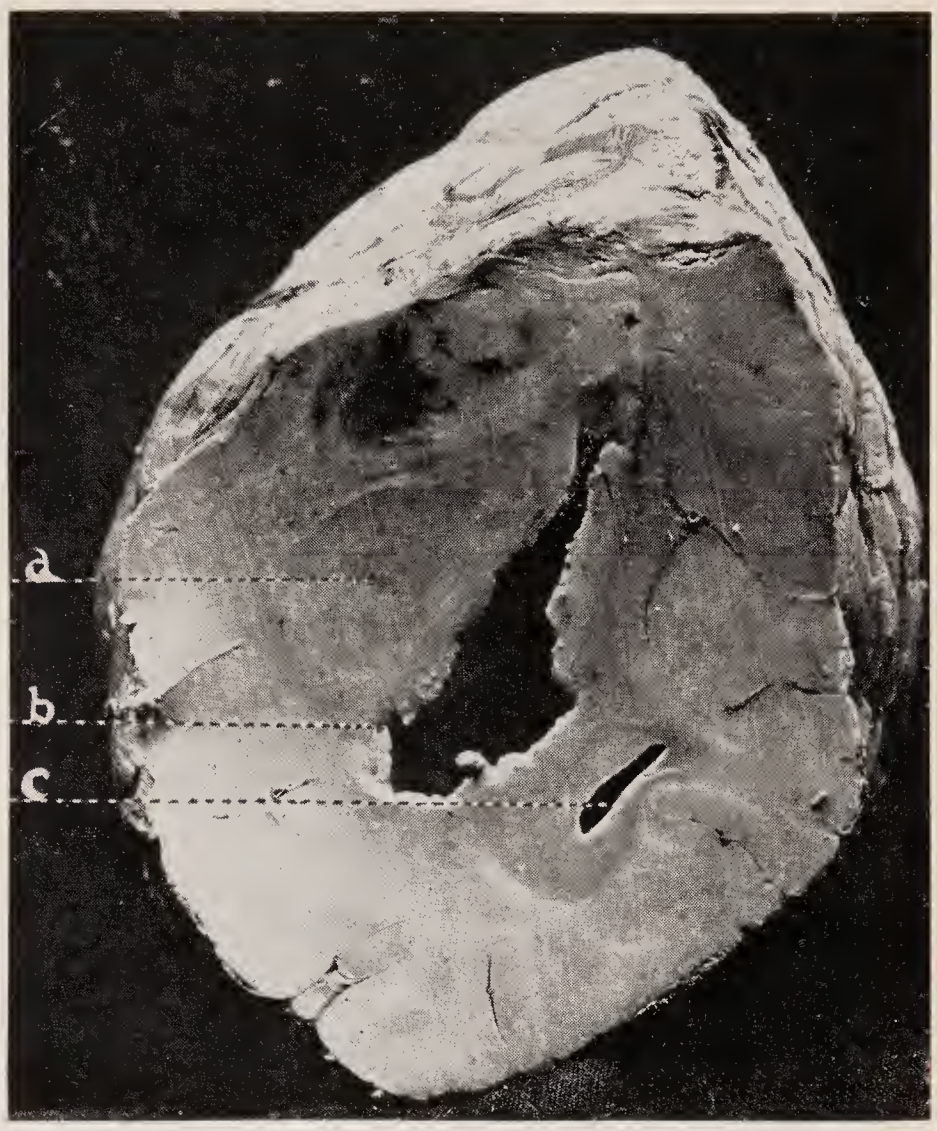

Fig. 27. D. F. A transverse section through occipital pole of brain. *a*, Primary abscess with firm wall; *b*, secondary abscess cavity; *c*, occipital pole of lateral ventricle.

mesoblastic elements of the blood vessels. In addition to the availability of the mesoblastic tissue, the quality of the resulting wall is likely to improve somewhat with the duration of the process. In our specimens, all of which were of less than a year's duration, the fibrous tissue proliferation reached a stage in no sense approximating the density of the wall shown under the heading of Type I. In Figure 20 the small band of fibrous tissue represented the most advanced stage of the fibrous tissue proliferation of an abscess wall which had existed as long as the wall shown in Figure 11. Hassin[1], however, described a wall of 8 years' duration in which the outer layer of the abscess wall was made up of adult fibrous tissue strands.

The question of time necessary for the proliferation of an abscess wall is an important one. It is certainly unusual for an abscess to exist for a period longer than a few months and walls of this type may be formed with great rapidity, the history of the abscess

[1] Hassin, G. B. Histopathological studies on brain abscess. Med. Rev., 1918, xciii, 91-96.

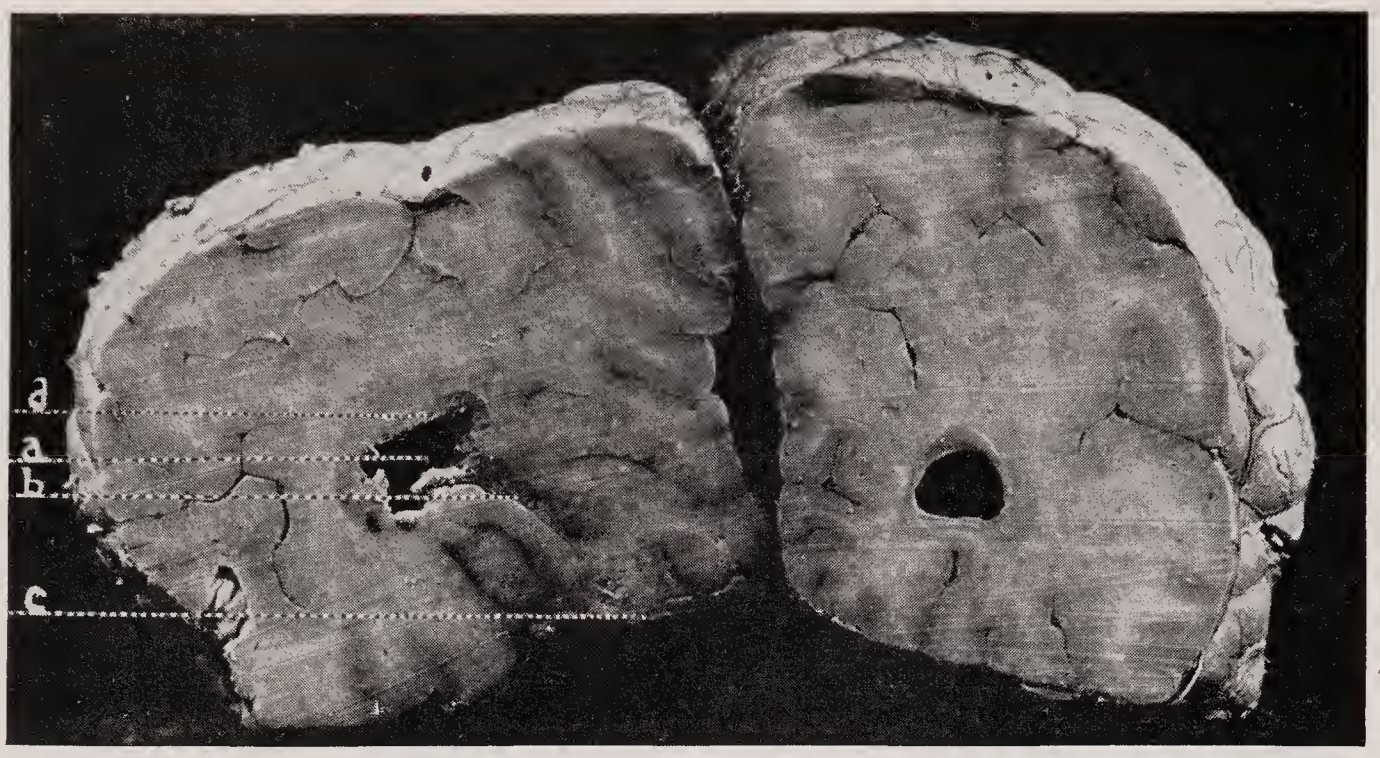

Fig. 28. P. V. Transverse section through the occipital pole of the brain shown in Figure 5. *a*, Occipital pole of the lateral ventricle converted into abscess cavity with necrotic wall; *b*, area of encephalitis, tract of evacuation of pus from the ventricle cavity to the subarachnoid space; *c*, thickened pia-arachnoid; *d*, site of section shown in Figure 29.

shown in Figure 18 indicating that the wall was formed within a period of 3 or 4 weeks. The hæmorrhages shown in Figure 19 were no doubt due to the very active vascular proliferation in the soft cerebral tissue.

Type III.—Walls of Varying Thickness, the Result of Glial Proliferation

Walls formed almost entirely of glial fibrils may be very heavy, but because of the delicate character of the fibrils the wall is not so resistant as one in which there is fibrous tissue. In Figure 21 the wall was visible macroscopically and in this picture of low magnification has the appearance of a thick, limiting membrane, but the delicate quality of the tissue is shown in Figure 22. The relative value of this type wall and the firm fibrous tissue wall is perhaps best shown in Figure 13, in which there is a firm fibrous tissue wall at *c* and at *d*, the adjacent cerebral tissue with glial proliferation. A photomicrograph of *d*, given in Figure 15, is similar to the abscess wall shown microscopically in Figure 22.

The neuroglial fibrils are again well shown in Figure 23, which was taken from the innermost part of the abscess wall. A large part of the cellular element has fallen out because of the necrosis, leaving the fibrils in plain view.

Type IV.—Walls Showing no Evidence of a Protective Reaction

Figure 24 shows an abscess which was the result of a virulent streptococcus planted deep in the substance of the cerebellar hemisphere. There is no evidence of a protective reaction and the lesion marks an intermediate stage between an encephalitis and the usual abscess formation, for though suppuration occurred there was no true barrier between the pus and the brain tissue. At *b* in Figure 24 there is an area of hæmorrhagic extravasation, the result of thrombosis, which is also well shown in Figure 25, the destructive process entirely replacing the usual proliferative reaction. The merely necrotic end-result of the destructive process is shown in Figure 26.

Abscesses of this type also occur as secondary lesions to firm wall abscesses. In Figure 27 the primary abscess has a thick wall, the building of which, no doubt, required several weeks, but the extension from this abscess was doubtless more recent due to escape of pus into the substance of the occipital lobe, an invasion altogether too sudden to allow the slowly proliferating glial tissue to form a protecting membrane.

D. F., age 29. Gunshot wound left hemisphere, July 14, 1918. Constant drainage of pus from the wound. Drainage of abscess, June 22, 1919. Death, June 30.

The architecture of the wall of the primary abscess is similar to that described under Group II. The firmness of the wall and heavy consistency of its content indicate a long duration. At *b*, however, is a larger abscess cavity with soft necrotic walls evidently due to a more recent extension from the original abscess.

Figure 28 shows an abscess, also the result of extension from the firm wall abscess, as seen in Figure 5. There was evidently leakage of pus into the occipital pole of the ventricle which was shut off anteriorly from the remaining part of the hemisphere ventricle so that the occipital pole was converted into an abscess cavity. Extending from the ventricle to the inferior surface of the brain, an inflammatory tract marks the site of the escape of pus into the subarachnoid space.

Figure 29 is a section from the ventricle wall of the specimen shown in Figure 28 illustrating the poor quality of the abscess wall, which consists only of necrotic brain tissue entirely incapable of acting as a barrier to the pus content in the cavity.

The escape of pus from the abscess into the ventricle is a very common method of termination of neglected abscesses, but the conversion of a portion of the ventricle into an abscess cavity as shown in Figure 26, is certainly an uncommon reaction. The formation of secondary abscesses may be due to the ineffectiveness of an abscess wall as a barrier to constantly accumulating pus, or to organisms, so that such extension is dependent upon the duration of the abscess and the virulence of the organism producing it.

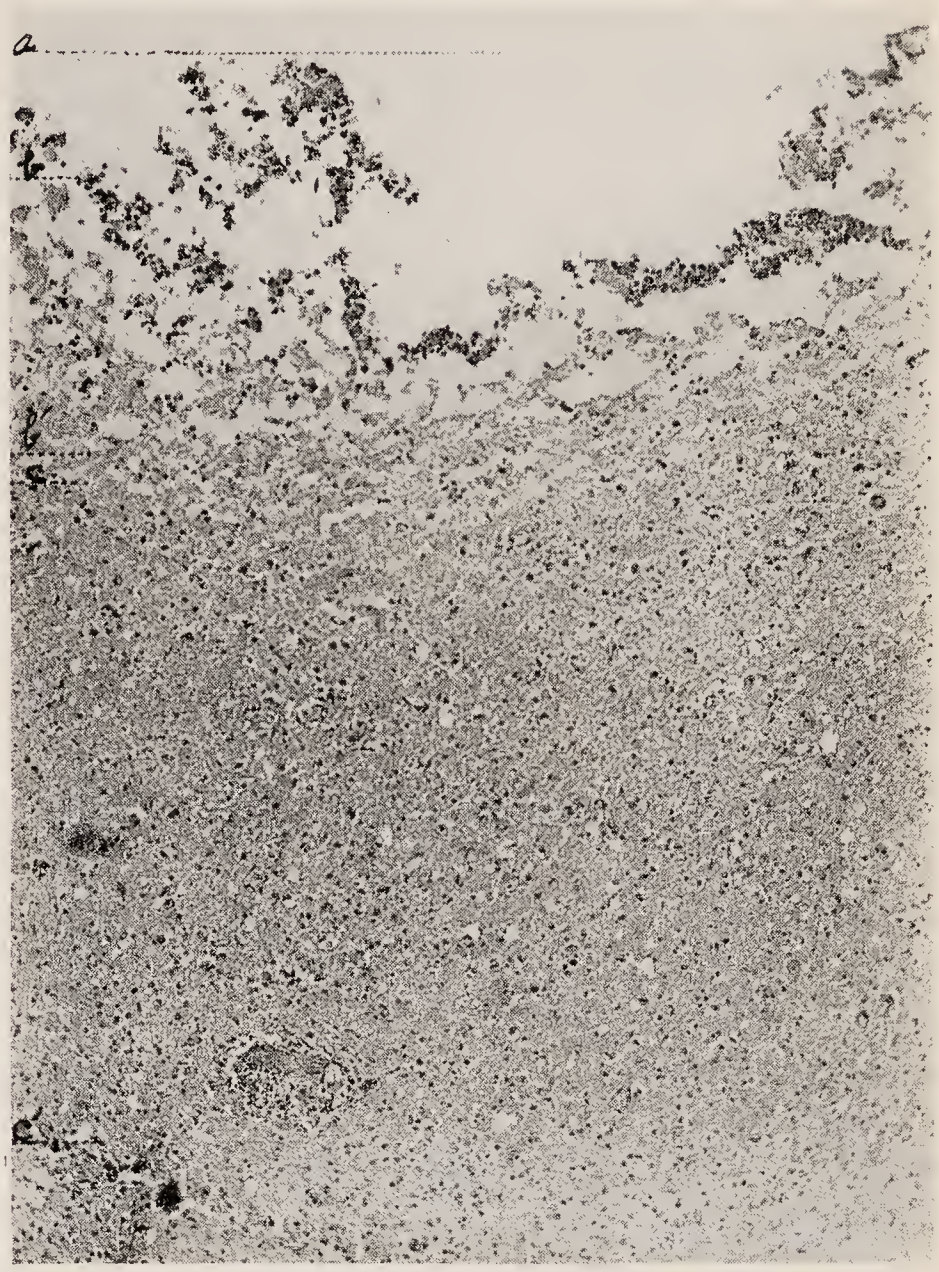

Fig. 29. P. V. Section of the abscess wall at *d* in Figure 28. *a*, Abscess cavity; *b* to b[1], necrotic brain tissue surrounding pus; *c* to *c*[1], brain tissue beyond the necrotic zone. ×85.

Summary

The clinical course of a brain abscess varies according to the infecting organism, the channel through which this organism reaches the brain, and the location of the infection in the brain substance as regards mesoblastic and epiblastic tissue.

This pathological study has been made as a basis for further consideration of the clinical data concerning these abscess cases.

Note.—This work has been done in the Neurological Laboratory of the Phipps Psychiatric Clinic, Johns Hopkins University, and has been greatly facilitated by the interest and co-operation of Dr. Adolf Meyer. I am much indebted, also, to Miss Cecilia Bisson for the histological and photographic preparation of the material.